HHS Public Access

Author manuscript

Environ Sci Technol. Author manuscript; available in PMC 2020 May 21.

Published in final edited form as:

Environ Sci Technol. 2019 May 21; 53(10): 6018–6024. doi:10.1021/acs.est.9b00471.

Polybrominated Diphenyl Ethers and Biphenyl in Serum: Time Trend Study from the National Health and Nutrition Examination Survey for Years 2005/06 through 2013/14

Andreas Sjödin[*], **Richard S. Jones**, **Lee-Yang Wong**, **Samuel P. Caudill**, and **Antonia M. Calafat**

Centers for Disease Control and Prevention, National Center for Environmental Health, Division of Laboratory Sciences, 4770 Buford Hwy, Atlanta Georgia 30341, United States

Abstract

Eleven polybrominated diphenyl ether (tri- to deca-BDE) congeners and 2,2′,4,4′,5,5′-hexabromobiphenyl (BB153) have been measured in pooled serum samples from the National Health and Nutrition Examination Survey (NHANES) for one decade (from survey years 2005/06 through 2013/14). The pools, which are representative of the general noninstitutionalized population of the United States, encompassed thirty-two demographic groups defined by sex, race/ethnicity (Mexican American, non-Hispanic black, non-Hispanic white, and all other race/ethnicities), and age (12–19, >20–39, >40–59, and ≥60 years). The adjusted geometric means were determined in a multiple linear regression model for the six congeners (BDE28, BDE47, BDE99, BDE100, BDE153, and BB153) with detectable concentrations in at least 60% of pools in each of the thirty-two demographic groups; the level of significance for all statistical comparisons thereof were determined. BDE154 and BDE209 were detected in 60% of the NHANES 2011/12 and 2013/14 pools; only these two survey periods were evaluated for these congeners. The percent change in concentration by a 2-year survey period was calculated. All examined PBDEs reported in five survey periods decreased in concentration, except BDE153, for which concentrations increased by 12.0% (95% CI 7.1–16.4) and 8.4% (95% CI 2.9–14.1) for the age groups 40–59 and ≥60 years, respectively; no significant change was observed in younger age groups. Excluding BDE153, we observed larger percentage decreases by a 2-year survey period for the age groups 12–19, 20–39, and ≥60 years compared with the age group 40−59 years. The percentage decrease by a two-year survey period ranged between −19.6% (BDE99, 20–39 years old) and −4.5% (BDE100, 40−59 years old). Although five polybrominated diphenyl ether (PDBE) congeners and BB153 are still frequently detected in the U.S. general population, PBDE concentrations have

[*]Corresponding Author: asjodin@cdc.gov.

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.9b00471.

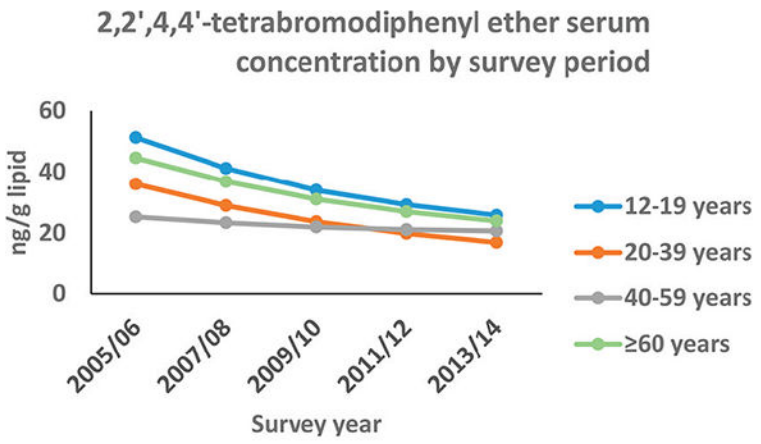
The number of serum pool measures by survey period; AGM concentration by demographic data for BDE28, BDE99, BDE100, BDE153, BDE154, BDE209, and BB153; total body fat concentration for NHANES 2005/06 by age and sex; AGM concentration by race/ethnic group; and AGM concentration by age group and survey period for BDE28 and BB153 (PDF)

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention (CDC). Use of trade names is for identification only and does not imply endorsement by the CDC, the Public Health Service, or the U.S. Department of Health and Human Services.

The authors declare no competing financial interest.

decreased since 2005–2006, likely, because of changes in manufacturing practices that started in the mid-2000s.

Graphical Abstract



INTRODUCTION

Polybrominated diphenyl ethers (PBDEs), used as flame retardants in consumer products and vehicles (e.g., cars, airplanes), were produced in three commercial mixtures defined by their average bromination degree, e.g., PentaBDE, OctaBDE, and DecaBDE.[1] PentaBDE was used as a flame retardant in polyurethane in applications such as furniture, seat cushions in vehicles, and in the polyurethane pads used under wall-to-wall carpets. The higher bromination degree products (Octa- and DecaBDE) were primarily used, among other applications, in hard plastics, including the casing for electrical appliances. Penta- and OctaBDE were withdrawn from the U.S. market in 2004, while commercial production of DecaBDE was discontinued in 2013, and any new imports of DecaBDE after 2013 would be subjected to significant new use reporting.[2]

The major exposure routes for PBDEs were diet and ingestion of residential dust with dust ingestion commonly considered to be most relevant.[3–6] Dietary ingestion includes consumption of fatty fish and other foods of animal origin, such as meat and poultry.[7] Diet is the dominating route of exposure for other persistent organic pollutants (POPs), such as polychlorinated biphenyls (PCBs), organochlorine pesticides, and polychlorinated dibenzo-*p*-dioxins and furans (PCDD/F), for the U.S. general population not occupationally exposed to these compounds. As time passes from the withdrawal of PBDEs from the U.S. market, and PBDEs are removed from the indoor environment by the replacement of furniture and consumer articles, dietary exposures are likely to become a more important route of human exposure.

The National Health and Nutrition Examination Survey (NHANES), administrated by the Centers for Disease Control and Prevention (CDC), provides an ongoing exposure assessment of the general noninstitutionalized population of the United States with respect to environmental pollutants and nutritional biomarkers by demographic characteristics, such

as age, sex, and race/ethnicity. One important objective of the NHANES biomonitoring program is to capture changes in chemical exposure (concentration in biological fluid, e.g., serum and/or urine), resulting from regulatory interventions, and changes in usage patterns related to human behavior and/or replacement chemicals reaching the market place. The National Reports on Human Exposure to Environmental Chemicals, which summarize the NHANES biomonitoring data,[8] include results on POPs, such as PCBs, organochlorine pesticides, PCDD/F, and PBDEs.

We report the changes in concentration of select PBDEs in NHANES serum pools representative of the general non- institutionalized U.S. population covering a decade (from 2005/06 through 2013/14).

MATERIALS AND METHODS

The CDC conducted NHANES continuously since 1999 (with biannual data releases) to assess the health and nutrition status of the civilian noninstitutional population.[8] The NHANES includes a physical examination, household interviews, collection of medical history, and serum and urine samples for measurement of clinical, environmental, and nutritional biomarkers.[8] Presented here are PBDEs and 2,2′,4,4′,5,5′- hexabromobiphenyl (BB153) measurements conducted on serum pools prepared by using individual sera collected from a one-third subset of all survey participants over the age of 12 years with a serum sample that had a sufficient volume for laboratory testing for NHANES 2005/06, 2007/08, 2009/10, 2011/12, and 2013/14. The Research Ethics Review Board of the National Center for Health Statistics (NCHS) reviewed and approved the study protocol. All participants gave informed written consent, and parents or guardians provided assent for participants younger than 18 years of age.

NHANES Sampling Weights.

The NHANES is a multistage, probability sampling survey. Oversampling of certain population subgroups was performed to increase the reliability and precision of estimates. Sampling weights were adjusted for unequal selection probability and were used to produce properly weighted population estimates of means, percentiles, and other descriptive statistics. The sample weights were determined by NCHS as follows. Nonpublic “base sample weights” were assigned to all persons solicited to participate in the survey and later adjusted for (1) nonresponse for interview and/or examination and (2) nonresponse for biological sampling. Subjects were selected from an approximately one- third subset of survey participants with a serum sample that had a sufficient volume for laboratory testing and were then assigned a subsample weight.

Pooling Strategy.

The pooling strategy was based on demographic groups that were defined by the cross classification of (1) sex (female and male), (2) age group (12–19, 20–39, 40–59, and ≥60 years), and (3) race/ethnicity. For NHANES 2005/06 and 2007/08, race/ethnicity groups were defined as Mexican American (MA), non- Hispanic black (NHB), non-Hispanic white (NHW), and all others (OTHER). For NHANES 2009/10, race/ethnicity was defined as MA,

NHB, NHW, other Hispanic (OH), and OTHER. NHANES 2011/12 and 2013/14 used the same grouping scheme as 2009/10, except that "OTHER" was divided further into non-Hispanic Asian (NHA) and the remaining. Thus, sample pooling was based on four race/ethnicity groups of MA, NHB, NHW, and OTHER (comprised of all race/ethnicity other than MA, NHB, and NHW).

The total number of available individual serum samples was 1973 (NHANES 2005/06), 2070 (NHANES 2007/09), 2322 (NHANES 2009/10), 1940 (NHANES 2011/12), and 2179 (NHANES 2013/14). These samples were used to create 247 (NHANES 2005/06), 264 (NHANES 2007/09), 301 (NHANES 2009/10), 251 (NHANES 2011/12), and 284 (NHANES 2013/14) serum pools, each comprised of eight individual samples. The number of pools created for each demographic group varied depending on the available number of individual samples (Supporting Information, Table S1).

We then assigned individual samples to pools using a SAS program (SAS 9.4; SAS Institute Inc., Cary, NC) to merge the (1) data file containing subsample weights, (2) data file containing demographic information, and (3) file containing the available serum volume by sample. The resulting data file was sorted by group and by the inverse subsample weight. A target of eight samples per pool was chosen based on the results of simulation experiments.[9] Then, for each demo- graphic group, the program assigned the first eight samples to the first pool, the next eight samples to the second pool, and so on until all samples were assigned to pools or until an insufficient number of serum samples were available to create additional pools. Thereafter, the program created a new variable, equal to the subsample weight of an individual sample divided by the sum of subsample weights of all samples in the corresponding pool, to calculate the proportional amount of each sample to be added to a pool so that the added volume was directly proportional to the subsample weight. The desired volume of each pool was 40 g; the program flagged any samples with insufficient available volume to meet the proportional volume requirement. The process was repeated after removal of flagged samples until no sample included in a pool had an insufficient volume. Measurements of pools created in this way are mathematically equal to weighted arithmetic means of the samples in the pools in keeping with the NHANES weighted sampling design, so that the estimates are representative of the general noninstitutionalized U.S. population.

Preparation of the Serum Pools.

The individual serum samples were thawed and mixed before the desired serum amounts were weighed into 60 mL Qorpak borosilicate glass bottles with Teflon PTFE-lined polypropylene screw caps (Qorpak, Bridgeville, PA). These vials were cleaned in a laboratory dish washer and heated overnight to 300 °C, and the caps were Soxhlet-extracted overnight in methanol to remove any contaminants. Serum was transferred using a new glass pipet for each sample. The total weight of each pool was determined as the difference in weight between the empty bottle and the bottle plus the serum on an AX105 (Mettler Toledo, Columbus, OH) balance.

Analytical Methods.

The sample preparation method was based on the original work by Hovander et al.[10] after modification and automation conducted in our laboratory.[11] The Standard Operating Procedure documentation is publicly available.[12] Two grams of serum was used for the measurements of PBDEs and BB153 randomly assigned to 24 sample batches for analysis; each analytical batch contained three quality control and three blank samples prepared with bovine serum (Gibco Inc. Grand Island, NY) diluted 1:40 with HPLC grade water (Labsolv Scientific Ltd., Tamil Nadu, India). All analytical data were subtracted from the median blank. The limits of detection (LODs) were determined as the higher of (1) three times the standard deviation of the amount present in blanks or (2) the instrumental LOD defined as the injected amount known to produce a gas chromatography/isotope dilution−high-resolution mass spectrometry signal/noise ratio > 10. The CV of the quality control samples made from the standard reference material 1958[13] was below 10%.

Statistical Methods.

We used SAS (version 9.4; SAS Institute Inc.; Cary, North Carolina) to perform statistical analyses. We conducted weighted multiple linear regression to examine the associations between the POP biomarkers and several variables (survey cycles (continuous), race (MA, NHW, NHB, OTHER for 5 survey periods covering the years 2005 through 2014), sex, age (12–19, 20–39, 40–59, and ≥60 years)). For the multiple regression models, we used the above variables and all their possible two-way interactions to calculate the adjusted geometric mean (AGM) concentrations. Because the distributions of the dependent variables' measurements were skewed to higher values, the dependent variable concentrations were $\log_{10}$ transformed in the multiple regression analyses. The final regression model included race/ethnicity, sex, and age as categorical main effects; survey period (1 through 5) as continuous main effects; and the interaction between the age and survey period. We also calculated the relative change in biomarker concentration associated with an unit increase in the survey cycle (per 2 years cycle, ng/lipid). For biomarker concentrations below the LOD, we imputed a value equal to the LOD divided by the square root of 2.[14] Statistical significance was set at $p < 0.05$.

We calculated the median and selected percentiles of total percent body fat by age and sex from publicly available data for NHANES 2005/06[15] after removing imputed data added to the data set (Table S9).

RESULTS

Eleven PBDE congeners (tri- to decaBDE) and BB-153 have been measured in NHANES 2005/06 through 2013/14. Only congeners with detection frequencies over 60% in all demographic groups were considered for the statistical analysis, i.e., BDE28 (90%), BDE47 (100%), BDE99 (99%), BDE100 (100%), BDE153 (100%), and BB153 (77%). BDE154 and BDE209 had detection frequencies over 60% only in NHANES 2011/12 and 2013/14; only these two surveys were included for BDE154 and BDE209. The overall detection frequency in NHANES 2011/12 and 2013/14 for BDE154 and BDE209 was 68 and 95%, respectively.

The AGM, 95% confidence interval (95% CI) and level of significance of the terms above when significant ($p < 0.05$) are given in Table 1 for BDE47 and Tables S1–S7 for BDE28, BDE99, BDE100, BDE153, BDE154, BDE209, and BB153. The level of significance for pairwise comparisons are also given in Tables 1 and S2–S8.

Change in PBDE Serum Concentration by Survey Period and Subjects Age.

The AGM and 95% CI of the serum concentrations of BDE28, BDE47, BDE99, BDE100, BDE153, and BB153 are given by age group and survey period (Figures 1 and S2–S8). The general trend is lower concentrations in more recent years with the distinct exception of BDE153 in the age groups 40–59 and ≥60 years, where we observed higher serum concentrations in more recent survey periods (Figure 1 and Table S2). The percentage change by the 2-year survey period in lipid-adjusted AGM serum concentration for BDE28, BDE47, BDE99, BDE100, BDE153, and BB153 is shown in Figure 2 and Table 2. All six PBDEs encompassing five survey periods decreased in concentration, except BDE153, which increased by 12.0% (95% CI 7.1–16.4) and 8.4% (95% CI 2.9–14.1) per the 2- year survey period for the age groups 40–59 years and ≥60, respectively. On the other hand, no significant change by the 2- year survey period was observed for BDE153 in the age groups of 12–19 and 20–39 year olds. Excluding BDE153, we observed larger percentage decreases by the 2-year survey period for the age groups of 12–19, 20–39, and ≥60 years compared with the age group of 40–59 years (Figure 2 and Table 2).

Concentration Difference by Sex.

The AGM concentrations by sex are given for BDE47, BDE99, BDE100, BDE153, and BB153 in Figure 3 and Tables 1 and S2–S8. Males had significantly ($p < 0.05$) higher lipid adjusted serum PBDE concentrations compared with females (range, 7 [BDE28] to 17% [BDE99]). BB153 was 40% higher in males compared to females ($p < 0.0001$).

AGM concentrations of BDE154 and BDE209, based on data for NHANES 2011/12 and 2013/14, also differed significantly by sex. BDE154 was 16% higher in males than females (Table S6). For BDE209, there was a significant interaction term between age and sex (Figure 4 and Table S7), where 12–19 year old males had lower concentrations than older males, while female concentrations did not vary significantly by age. Males had higher serum concentrations at 20–39 (37%), 40–59 (45%), and ≥60 (18%) years than females, and there were no significant differences for the 12–19 year olds by sex.

Concentration Difference by Ethnicity/Race.

NHB had 16–20% higher BDE47 concentration ($p < 0.01$) than MA, NHW, and OTHER (Table 1 and Figure S1). BDE99 and BDE100 concentrations were also higher in NHB than in other race/ethnic groups ($p < 0.01$). In the case of BDE153, no statistical differences were observed among MA, MHB, and NHW, while the OTHER group had lower serum concentrations compared to MA and NHW ($p < 0.001$).

DISCUSSION

The fact that five PBDE congeners and BB153 had detection frequencies of 77% and above suggests that these POPs continue to be detectable in a large proportion of the U.S. population years to decades after discontinuation of their sale and registered use. However, the continuous presence of these POPs is not unexpected, considering that PBDEs are persistent and still present in upholstered furniture and polyurethane pads under wall−wall carpets manufactured before the phase- out.[2] Further, because the pad under wall-to-wall carpets can also contain recycled polyurethane, PBDEs might have been introduced into homes after the phase-out of these compounds. On the other hand, although BB153 has not been commercially produced in the United States since the 1970s, its biological half-life, estimated to be 11 years,[16] can explain the detection of this substance in recently collected human serum.

BDE209 was not included in previous publications on NHANES data from our laboratory[17–19] because of its overall relatively low detection frequency. In recent years, we were able to reduce the analytical background with the concomitant decrease in the LOD (i.e., to 0.8 ng/g lipid or a factor of 3 lower in NHANES 2011/12 and 2013/14 than in previous surveys for a 2-g serum sample). Of interest, the BDE209 serum concentration is relatively low compared to other BDEs (e.g., BDE47), even though BDE209 is present at much higher (4-fold) concentrations in indoor dust than BDE47.[5] This lower serum concentration is most likely due to a relative short biological half-life of BDE209, corresponding to 2 weeks in humans.[20] The biological half-life of lower bromination degree BDEs is longer and measured in years; however, these estimates are modeled and not directly observed after a substantial reduction in exposure and are hence associated with substantial uncertainty and/or bias.[21,22]

The lower concentration of PBDEs in the age group of 40–59 year vs younger and older subjects[17–19] has been puzzling. At this point, with data from multiple NHANES cycles, we further see that the overall decrease over time in serum PBDEs is lower in the same age group (40–59 year olds) for all PBDEs investigated. Further, the hexabrominated congener BDE153 is increasing in concentration in the 40–59 year olds and, to a lesser extent, in the group of 60 years of age and older. It is not plausible that exposure would have increased for the 40–59 year olds but not for other age groups. It is possible that BDE153 could be formed from higher bromination degree congeners, e.g., BDE209. However, because we did not detect other congeners/degradation products with bromination degrees between BDE209 and BDE153 that are not present in technical products, such an explanation is plausible but unlikely. Because PBDEs are lipophilic and primarily distribute into the lipid rich tissues of the body, a more likely explanation is that increases in body weight with age led to a successively larger dilution of PBDEs in an increasingly fat distribution volume (Table S9). The total body fat percentage is 19 and 26% higher in the 49–59 year age group vs the 12–19 year olds for women and men, respectively. Hence, a change in body fat content may explain overall lower concentration at an older age but cannot alone explain the increase in BDE153 concentration observed in the older age group. A smaller decrease or increase of the PBDE concentrations by the survey period among the 40–59 year olds may, on the other hand, relate to increased frequency of obesity in 40–59 year olds. With the larger distribution

volume of PBDEs in obese persons, a smaller proportion of PBDEs would circulate in serum where metabolism and transport to metabolism organs may occur. Therefore, an overall increase in the PBDE biological half-life is possible in subjects who are obese, as shown for the anxiety drug Diazepam, another lipophilic compound.[23] The observed increase in BDE153 concentration by the survey period in the age groups of 40–59 and ≥60 years of age may hence occur because of an increase in biological half-life resulting in a greater intake than elimination in these age groups. Unfortunately, no biological half-lives are known for PBDEs, except for BDE183 (3 months) and BDE209 (2 weeks).[20]

Excluding BDE209, we observed overall lower concentrations from 7 (BDE28) to 27% (BDE153) in females vs males. This sex difference is likely related to differences in whole body fat content causing differences in distribution volume for men and women for these lipophilic compounds. On the other hand, for BDE209, we observed significantly higher concentrations in males compared with females for the age groups of 20–39, 40–59, and ≥60 years; however, the reason for such a sex by age difference is unknown.

We observed higher concentration in NHB subjects for BDE47, BDE99, and BDE100 compared with MA, NHW, and OTHER, while BDE153 was higher than in the other race/ethic groups for MHW. These marginal differences may be related, at least in part, to occupational exposures, among other causes.

Of interest, BDE47 concentrations in NHANES 2013/14 were about an order of magnitude higher than the peak concentrations of BDE47 in the Swedish general population (approximately 2 ng/g lipid) in 1998.[24] Assuming that the concentration of PBDEs will decrease between 5 and 20% every two years in the United States, PBDE concentrations will continue to exceed the peak concentrations observed in other parts of the world. Future monitoring efforts at the CDC will include analyzing individual serum samples from NHANES 2017/18 to track lifestyle factors associated with body burden of PBDEs, such as age of primary residence, socioeconomic status, and BMI.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGMENTS

The authors would like to acknowledge the researchers who made this work possible. Emily McDonald for preparing all samples for gas chromatography/high resolution mass spectrometry (HRMS) analysis; Sarah Anderson, Darlinda Harry, Carolyn Hodge, Wanda Whitfield, and Yalin Zhang for HRMS analysis; Troy Cash, Yolanda Dalton, and Gerard Dublin for preparing the pools; Patricia McClure and Michelle Moore for data handling; and Pamela Olive for the serum lipid measurements. We also would like to acknowledge NCHS for serum collection and study administration.

REFERENCES

(1). Alaee M; Arias P; Sjodin A; Bergman A An overview of commercially used brominated flame retardants, their applications, their use patterns in different countries/regions and possible modes of release. Environ. Int 2003, 29 (6), 683–9. [PubMed: 12850087]

(2). Polybrominated Diphenyl Ethers (PBDEs), Assessing and Managing Chemicals under TSCA; U.S. Environmental Protection Agency: Washington, D., 2017 https://www.epa.gov/assessing-and-

managing-chemicals-under-tsca/polybrominated-diphenyl-ethers-pbdes (accessed October 18, 2018).

(3). Jones-Otazo H; Clarke J; Diamond M; Archbold J; Ferguson G; Harner T; Richardson G; Ryan J; Wilford B Is house dust the missing exposure pathway for PBDEs? An analysis of the urban fate and human exposure to PBDEs. Environ. Sci. Technol 2005, 39 (14), 5121–5130. [PubMed: 16082939]

(4). Lorber M Exposure of Americans to polybrominated diphenyl ethers. J. Exposure Sci. Environ. Epidemiol 2008, 18 (1), 2–19.

(5). Sjodin A; Papke O; McGahee E; Focant JF; Jones RS; Pless-Mulloli T; Toms LM; Herrmann T; Muller J; Needham LL; Patterson DG Jr. Concentration of polybrominated diphenyl ethers (PBDEs) in household dust from various countries. Chemo-sphere 2008, 73 (1), S131–S136.

(6). Johnson-Restrepo B; Kannan K An assessment of sources and pathways of human exposure to polybrominated diphenyl ethers in the United States. Chemosphere 2009, 76 (76), 542–548. [PubMed: 19349061]

(7). Schecter A; Haffner D; Colacino J; Patel K; Päpke O; Opel M; Birnbaum L Polybrominated Diphenyl Ethers (PBDEs) and Hexabromocyclodecane (HBCD) in Composite U.S. Food Samples. Environ. Health Perspect 2010, 118 (3), 357–362. [PubMed: 20064778]

(8). National Report on Human Exposure to Environmental Chemicals; National Center for Environmental Health (NCEH), Centers for Disease Control and Prevention (CDC) https://www.cdc.gov/exposurereport/index.html (accessed October 18, 2018),.

(9). Caudill S Characterizing populations of individuals using pooled samples. J. Exposure Sci. Environ. Epidemiol 2010, 20 (1), 29–37.

(10). Hovander L; Athanasiadou M; Asplund L; Jensen S; Klasson Wehler E Extraction and cleanup methods for analysis of phenol and neutral organohalogens in plasma. J. Anal. Toxicol 2000, 24, 696–703. [PubMed: 11110024]

(11). Jones R; Edenfield E; Anderson S; Zhang Y; Sjodin A Semi-automated extraction and cleanup method for measuring persistent organic polutants in human serum. Organohalogen Comp 2012, 74, 97–98.

(12). Sjodin A Laboratory Procedure Manual: Polybrominated diphenyl ethers (PBDEs), Polybrominated Biphenyls (PBBs), Polychlorinated biphenyls and Persistent Pesticides (PPs). https://wwwn.cdc.gov/nchs/data/nhanes/2013-2014/labmethods/PCBPOL_H_MET.pdf (accessed October 18, 2018),.

(13). Schantz MM; Eppe G; Focant JF; Hamilton C; Heckert NA; Heltsley RM; Hoover D; Keller JM; Leigh SD; Patterson DG Jr.; Pintar AL; Sharpless KE; Sjodin A; Turner WE; Vander Pol SS; Wise SA Milk and serum standard reference materials for monitoring organic contaminants in human samples. Anal. Bioanal. Chem 2013, 405 (4), 1203–11. [PubMed: 23132544]

(14). Hornung R; Reed LD Estimation of average concentration in the presence of nondetectable values. Appl. Occup. Environ. Hyg 1990, 5 (1), 46–51.

(15). National Health and Nutrition Examination Survey 2005−2006 Data Documentation, Codebook, and Frequencies; National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (CDC), 2007.

(16). Rosen DH; Flanders WD; Friede A; Humphrey HEB; Sinks TH Half-life of polybrominated niphenyl in human sera. Environ. Health Perspect 1995, 103 (3), 272–274. [PubMed: 7768229]

(17). Sjodin A; Wong LY; Jones RS; Park A; Zhang Y; Hodge C; Dipietro E; McClure C; Turner W; Needham LL; Patterson DG Jr. Serum concentrations of polybrominated diphenyl ethers (PBDEs) and polybrominated biphenyl (PBB) in the United States population: 2003−2004. Environ. Sci. Technol 2008, 42 (4), 1377–84. [PubMed: 18351120]

(18). Sjodin A; Jones RS; Caudill SP; Wong LY; Turner WE; Calafat AM Polybrominated Diphenyl Ethers and Other Persistent Organic Pollutants in Serum Pools from the National Health and Nutrition Examination Survey: 2001−2002. Environ. Sci. Technol. Lett 2014, 1 (1), 92–96. [PubMed: 27933302]

(19). Sjodin A; Jones RS; Caudill SP; Wong LY; Turner WE; Calafat AM Polybrominated diphenyl ethers, polychlorinated biphenyls, and persistent pesticides in serum from the national health and

nutrition examination survey: 2003−2008. Environ. Sci. Technol 2014, 48 (1), 753–60. [PubMed: 24298999]

(20). Thuresson K; Hoglund P; Hagmar L; Sjodin A; Bergman A; Jakobsson K Apparent half-lives of hepta- to decabrominated diphenyl ethers in human serum as determined in occupationally exposed workers. Environ. Health Perspect 2006, 114 (2), 176–81. [PubMed: 16451851]

(21). Geyer HJ; Schramm K-W; Darnerud PO; Aune M; Anton Feicht E; Fried KW; Henkelmann B; Lenoir D; Schmid P; McDonald TA Terminal elimination half-lives of the brominated flame retardants TBBPA, HBCD, and lower brominated PBDEs in humans. Organohalogen Comp 2002, 66, 3820–3825.

(22). Wong F; Cousins IT; MacLeod M Bounding uncertainties in intrinsic human elimination half-lives and intake of polybrominated diphenyl ethers in the North American population. Environ. Int 2013, 59, 168–174. [PubMed: 23831542]

(23). Abernethy DR; Greenblatt DJ; Divoll M; Shader RI Prolonged accumulation of Diazepam in obesity. J. Clin. Pharmacol 1983, 23, 369–376. [PubMed: 6415130]

(24). Meironyté Guvenius D Organohalogen contaminants in humans with emphasis on polybrominated diphenyl ethers; Karolinska Institutet: Stockholm, Sweden, 2002.

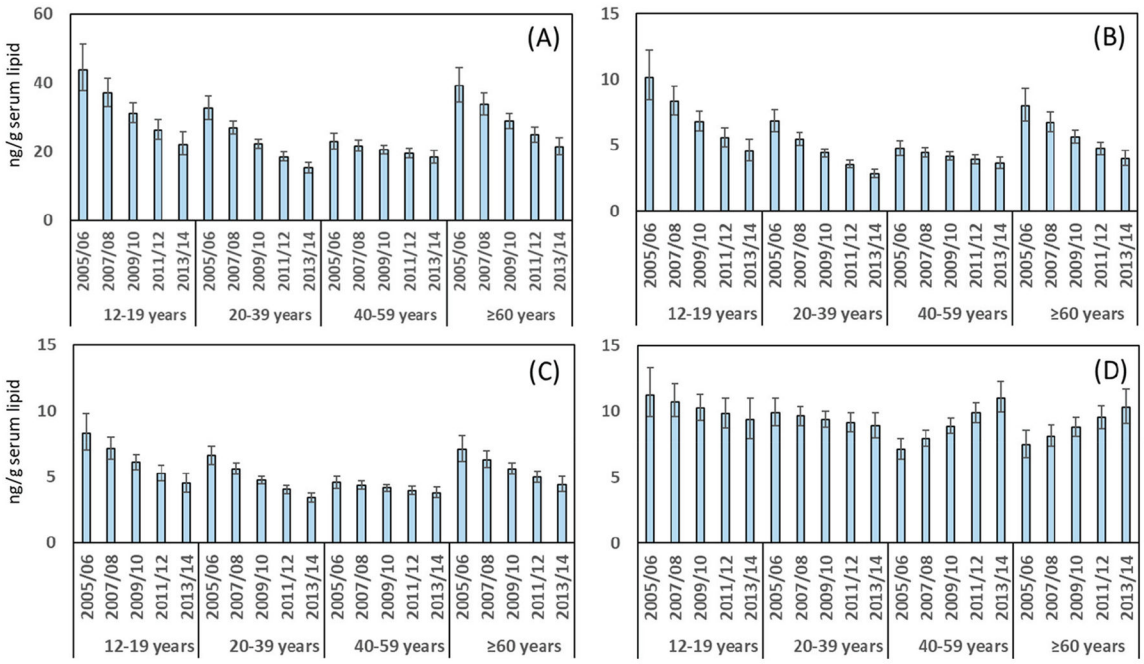


Figure 1.

Adjusted geometric mean concentration (ng/g lipid) by age group and survey period for BDE47 (A), BDE99 (B), BDE100 (C), and BDE153 (D). Bars indicate 95% CIs.

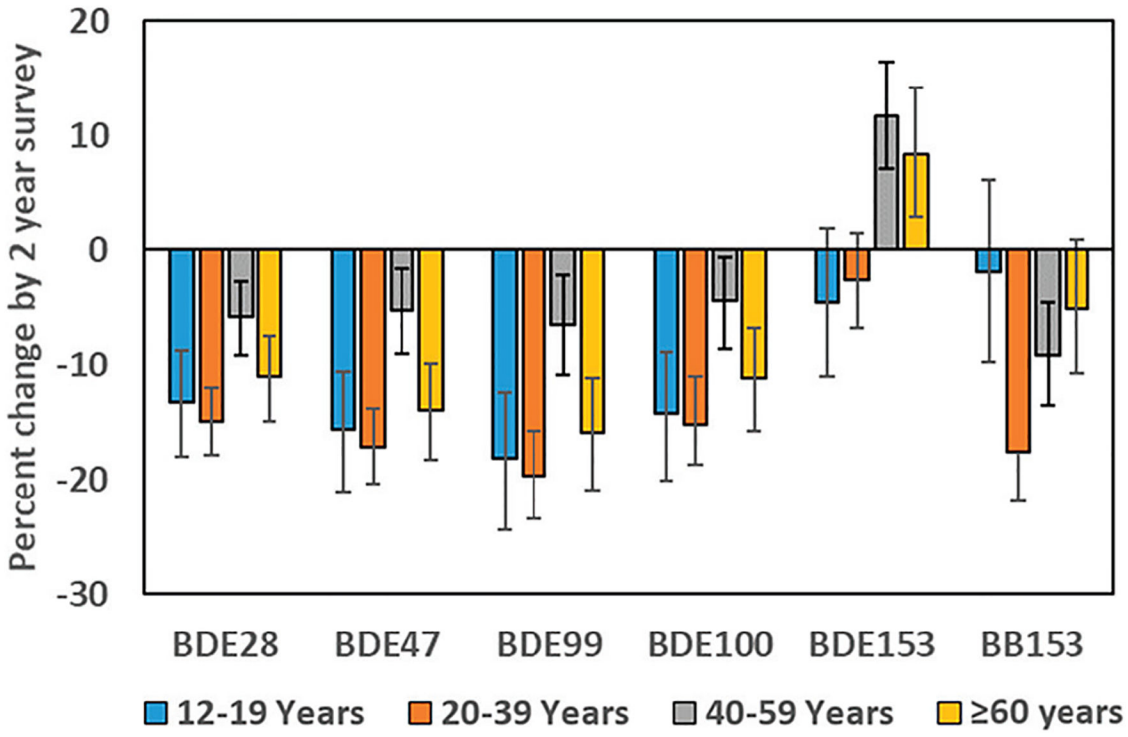


Figure 2.
Percent change in serum concentration by the 2-year survey cycle.

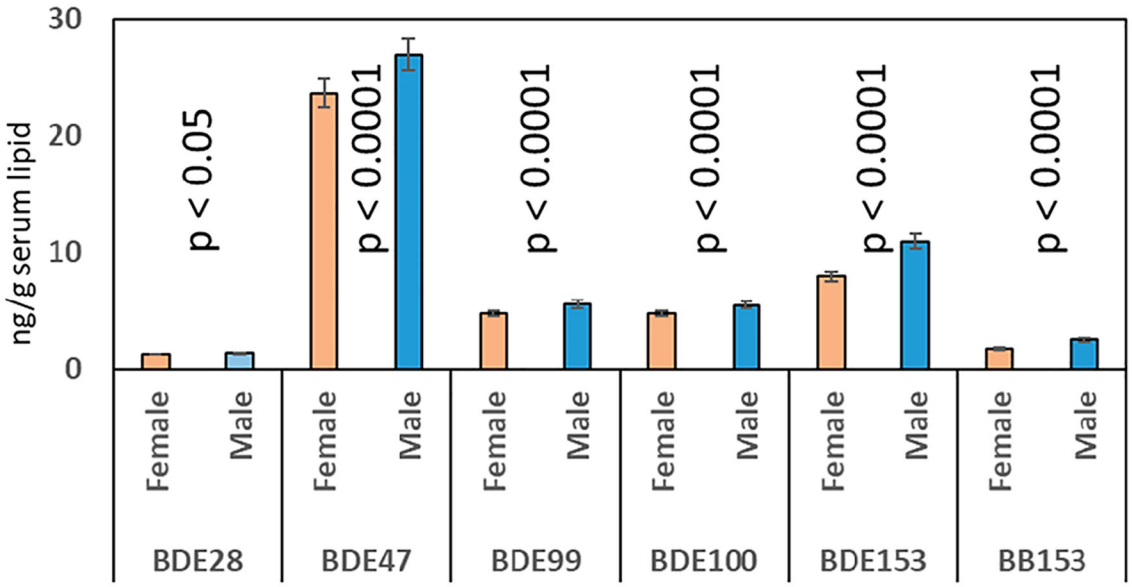


Figure 3.
Adjusted geometric mean PBDE concentration (ng/g lipid) by sex.

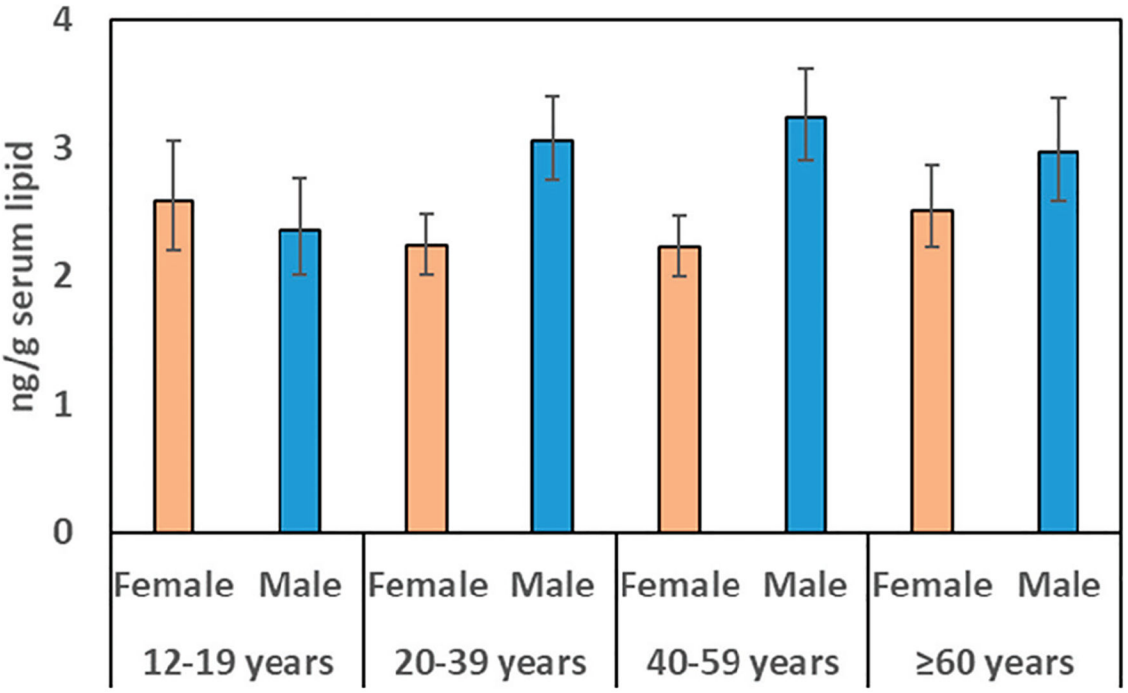


Figure 4.

BDE-209 adjusted geometric mean concentration (ng/g lipid) by age group and sex. Bars indicate 95% CI. BDE209 concentration in males 12–19 years are significantly lower than other male age groups.

Table 1.

AGM (ng/g Lipid) and 95% CI by Demographic Groups for 2,2′,4,4′-Tetrabromodiphenyl Ether (BDE47)

category	AGM	95% CI	pairwise comparison: level of significance (*p*)			
Race/Ethnicity($p < 0.01$)			MA	NHB	NHW	
MA	23.3	(21.7–25.1)				
NHB	29.2	(26.4–32.3)	<0.001			
NHW	24.6	(23.5–25.7)	0.2	<0.01		
OTHER	24.1	(22.1–26.3)	0.6	<0.01	0.7	
Sex($p < 0.0001$)						
female	23.6	(22.4–24.9)	<0.0001			
male	26.9	(25.5–28.4)				
Age and Survey (Interaction Term, $p < 0.0001$)			12–19 years	20–39 years	40–59 years	≥60 years
NHANES 2005/06						
12–19 years	43.8	(37.6–51.2)				
20–39 years	32.6	(29.4–36.0)	<0.01			
40–59 years	22.9	(20.7–25.4)	<0.0001	<0.0001		
≥60 years	39.1	(34.4–44.5)	0.3	<0.05	<0.0001	
NHANES 2007/08						
12–19 years	37.0	(33.1–41.3)				
20–39 years	27.0	(25.1–29.9)	<0.0001			
40–59 years	21.7	(20.2–23.4)	<0.0001	<0.0001		
≥60 years	33.6	(30.6–36.9)	0.2	<0.001	<0.0001	
NHANES 2009/10						
12–19 years	31.2	(28.5–34.1)				
20–39 years	22.3	(21.1–23.7)	<0.0001			
40–59 years	20.6	(19.4–21.9)	<0.0001	<0.05		
≥60 years	28.9	(26.8–31.2)	0.2	<0.0001	<0.0001	
NHANES 2011/12						
12–19 years	26.3	(23.6–29.3)				
20–39 years	18.5	(17.2–19.9)	<0.0001			
40–59 years	19.5	(18.2–21.0)	<0.0001	0.3		
≥60 years	24.9	(22.8–27.1)	0.4	<0.0001	<0.0001	

category	AGM	95% CI	pairwise comparison: level of significance (*p*)			
NHANES 2013/14						
12–19 years	22.2	(19.0–25.9)				
20–39 years	15.3	(13.9–16.9)	<0.001			
40–59 years	18.5	(16.7–20.4)	<0.05	<0.01		
≥60 years	21.4	(19.0–24.1)	0.7	<0.0001	0.06	

Table 2.

Percent Change in PBDE and 2,2′,4,4′,5,5′-Hexabromobiphenyl Concentration by the 2-Year Survey Period

	percent change in concentration (ng/g lipid) by the 2-year survey period with 95% CI[a]							
compound	**12–19 years**	***p*-value**	**20–39 years**	***p*-value**	**40–59 years**	***p*-value**	**>60 years**	**p-value**
BDE28	−13.3 (−17.8/−8.5)	<0.0001	−15.0 (−17.9/−12.0)	<0.0001	−5.9 (−9.0/−2.6)	<0.001	−11.1 (−14.8/−7.2)	<0.0001
BDE47	−15.7 (−20.7/−10.3)	<0.0001	−17.2 (−20.4/−13.8)	<0.0001	−5.3 (−8.9/−1.5)	<0.01	−14.0 (−18.1/−9.7)	<0.0001
BDE99	−18.2 (−24.0/−12)	<0.0001	−19.6 (−23.3/−15.8)	<0.0001	−6.4 (−10.7/−2.0)	<0.01	−15.9 (−20.6/−10.9)	<0.0001
BDE100	−14.3 (−19.7/−8.5)	<0.0001	−15.2 (−18.7/−11.6)	<0.0001	−4.5 (−8.3/−0.4)	<0.05	−11.2 (−15.6/−6.5)	<0.0001
BDE153	−4.6 (−10.6/1.9)	0.16	−2.6 (−6.6/1.6)	0.22	12.0 (7.1/16.4)	<0.0001	8.4 (2.9/14.1)	<0.01
BB153	−1.9 (−9.2/6.1)	0.6	−17.6 (−21.6/−13.3)	<0.0001	−9.2 (−13.6/−4.5)	<0.001	−5.2 (−10.8/0.9)	0.14

[a]Percent change = $100 \times (10^{\beta} - 1)$, where β is the estimated model coefficient for the variable 2-year survey.